the mean (±1 SD) Pb concentrations between minorities of 57 ng/m^3 (±24 ng/m^3) and nonminorities of 22 ng/m^3 (±3.4 ng/m^3) in personal air exposures, although the differences were not significant (p=0.147). Similarly, differences were noted between minorities (26±12 ng/m^3) and nonminorities (13±2.6 ng/m^3) in indoor air, although these were also not significantly different (p=0.266). When the age of the home was considered in the analysis, it was found that Pb concentrations were significantly (p=0.036) higher in homes built before 1940 than in homes built between 1960 and 1979, with mean (±1 SD) values of 46 ng/m^3 (±1.6 ng/m^3) and 13 ng/m^3 (±2.1 ng/m^3), respectively. The Pb concentrations measured in indoor air in homes built before 1940 were not significantly different from mean (±1 SD) Pb concentrations of 22 ng/m^3 (±5.1 ng/m^3) and 23 ng/m^3 (±5.1 ng/m^3) measured in indoor air in homes built between 1940 and 1959 and between 1980 and 1995, respectively.

5.5.2 Water

Pb has been monitored in surface water, groundwater, and drinking water throughout the United States and other countries. The concentration of Pb in surface water is highly variable depending upon sources of pollution, Pb content of sediments, and characteristics of the system (pH, temperature, etc.). Levels of Pb in surface water and groundwater throughout the United States typically range between 5 and 30 µg/L, although levels as high as 890 µg/L have been measured (EPA 1986a). Mean levels of Pb in surface water measured at 50,000 surface water stations throughout the United States are 3.9 µg/L (based on 39,490 occurrences) (Eckel and Jacob 1988). The median Pb level in natural river water is 5 µg/L, with a range of 0.6–120 µg/L (Bowen 1966). Pb levels in seawater are estimated as 0.005 µg/L (EPA 1982c). Pb concentrations in surface water are higher in urban areas than in rural areas (EPA 1982c). Using the EPA Storage and Retrieval (STORET) database, from January 1, 2005 to May 16, 2005, Pb had been detected in surface water in Washington, Utah at concentrations of 20.5 and 142 µg/L and surface water from Salt Lake City, Utah at 7.75 µg/L (EPA 2005b). Pb was not detected above the detection limits in 224 other surface water samples obtained from various locations in Utah and Iowa over the sampling period (EPA 2005b).

Urban storm water runoff is an important source of Pb entering receiving waterways. Pb is found in building material (brick, concrete, painted and unpainted wood, roofing, and vinyl), and automotive sources (brakes, used oil), which contribute to runoff (Davis et al. 2001). The largest contributing sources were siding and roofing. Soto-Jiménez-Flegal (2009) evaluated the sources of Pb pollution in the Gulf of California, northwest Mexico by sampling urban and rural areas for Pb levels and isotope ratios. Urban street dust (157 µg/g), agricultural soils (29.0 µg/g), and surface estuary sediments (35.6 µg/g) were all

higher than natural bedrock (16.0 μg/g). Isotopic ratios in rural and soil runoff samples were comparable to natural Pb containing bedrock. Pb concentrations in the suspended particulate matter were measured in sewage effluent (132 μg/g), agricultural effluent (29.3 μg/g), river runoff (7.3 μg/g), and estuary water (68.3 μg/g). Urban, street dust, and sewage showed contributions from automotive emissions from past leaded gasoline combustion.

Based on a survey of 900 public water supply systems, EPA (1988b) estimated that 99% of the 219 million people in the United States using public water supplies are exposed to drinking water with levels of Pb <5 μg/L and approximately 2 million people are served by drinking water with levels of Pb >5 μg/L. A survey of 580 cities in 47 states indicated that the national mean concentration of Pb in drinking water was 29 μg/L after a 30-second flushing period (EPA 1986a, 1989e); however, it was estimated that the average Pb content of drinking water in 1988 decreased to 17 μg/L (Cohen 1988). In 1986, the Safe Drinking Water Act Amendments banned the use of Pb solder or flux containing >0.2% Pb and the use of Pb pipes or fittings that contained >8% Pb (EPA 1986a, 1989e).

EPA (1991b) examined the occurrences of Pb in source water and distributed water. By resampling at the entry point to the distribution system, few samples were found to contain Pb at levels above 5 μg/L. EPA estimated that approximately 600 groundwater systems may have water leaving the treatment plant with Pb levels above 5 μg/L. Based on several data sets, it was estimated that <1% of the public water systems in the United States have water entering the distribution system with Pb levels above 5 μg/L. These systems are estimated to serve <3% of the population that receives drinking water from public systems (EPA 1991b). Analyses done in support of the EPA Lead and Copper Rule estimated that, over the period 2003–2005, <2% of public water systems exceeded the Pb action level of 15 μg/L (EPA 2007a).

Pb levels ranging between 10 and 30 μg/L can be found in drinking water from households, schools, and office buildings as a result of plumbing corrosion and subsequent leaching of Pb. The combination of corrosive water and Pb pipes or Pb-soldered joints in either the distribution system or individual houses can create localized zones of high Pb concentrations that exceed 500 μg/L (EPA 1989d, 2007a).

Quantitative data on the nationwide range of Pb levels in drinking water drawn from the tap (which would include Pb corrosion byproduct) were insufficient to assign a national value at the time of the EPA (1991b) publication. One set of data comprised of 782 samples taken in 58 cities in 47 states shows that the average Pb level in tap water was 13 μg/L with 90% of the values below 33 μg/L (EPA 1991b). In the NHEXAS study that was conducted during 1995–1996, Pb concentrations were measured in tap drinking

water (flushed for 15 minutes) taken from 82 homes in Arizona (O'Rourke et al. 1999), 441–444 homes in EPA Region V (Thomas et al. 1999), and 381 homes in Maryland (Ryan et al. 2000). Median Pb concentrations of 0.4, 0.37, and 0.33 µg/L were determined in the Arizona, EPA Region V, and Maryland regional studies, respectively. Mean values (±1 SD) of 0.84 µg/L (±1.8 µg/L) and 1.08 µg/L (±2.01 µg/L) were calculated for the EPA Region V and Maryland studies, respectively, and are much lower than the mean concentrations of Pb in drinking water determined in previous EPA estimates.

Pb levels are also known to increase when tap water is heated in boiling kettles that contain Pb in their heating elements. Pb concentrations in tap water were found to vary depending on the age of nine homes in New Jersey. In homes built in the 1980s, median Pb concentrations in the first-draw sample were higher (17.9 µg/L) than in first-draw samples (1.86 µg/L) taken from homes built in the 1970s (Murphy and Hall 2000). Leaching of Pb from kitchen plumbing fixtures was given as the reason for the high Pb concentrations in the first-draw samples. An additional water draw (>2 L) found decreased Pb concentrations in tap water for all homes. However, the median concentration of Pb in samples taken from homes built in the 1980s was higher (2.45 µg/L) than in samples taken from 1970s homes (0.14 µg/L). The Pb concentrations in these higher volume samples are attributed to Pb leaching from solder joints in basement piping and the water meter on the public water service line that may be more prevalent in the more recently built homes.

5.5.3 Sediment and Soil

Sediments contain considerably higher levels of Pb than corresponding surface waters. Concentrations of Pb in river sediments have been estimated at about 23 mg/kg (EPA 1982c; Fitchko and Hutchinson 1975), and concentrations of Pb in coastal sediments range from 1 to 912 mg/kg with a mean value of 87 mg/kg (EPA 1982c; Nriagu 1978). Data from the STORET (1973–1979) database of Eastern and Midwestern U.S. river basins indicates maximum Pb concentrations in river sediments of 440–1,000 mg/kg and mean Pb concentrations of 27–267 mg/kg (EPA 1982c). More current data obtained from the EPA STORET database (from January 1, 2004 to May 16, 2005) showed that Pb has been detected in sediment samples from Honolulu, Hawaii (0.75–6.2 mg/kg), various locations of South Carolina (<1–21 mg/kg), Dade County, Florida (4.7–17.9 mg/kg), and various locations in Tennessee (6–50 mg/kg) (EPA 2005c). Surface sediment concentrations in Puget Sound ranged from 13 to 53 mg/kg (Bloom and Crecelius 1987). An analysis of sediments taken from 10 lakes in Pennsylvania indicated that the elevated Pb values were not derived from leaching of Pb from the native rocks as a result of acid deposition, but rather originated from anthropogenic Pb deposition (probably from automotive emissions) on the soil surface

and subsequent runoff of soil particulates into the lake (Case et al. 1989). Local sources of Pb releases can also contribute significantly to Pb content in sediments (Gale et al. 2004). For example, Pb concentrations in sediments located near mines and or sites containing mine tailings in the old Pb belt of Missouri were greatly elevated, 10,550–12,400 mg/kg sediment (dry weight) compared to unaffected sediments (72–400 mg/kg dry weight) (Gale et al. 2002).

The natural Pb content of soil derived from crustal rock, mostly as galena (PbS), typically ranges from <10 to 30 μg/g soil. However, the concentration of Pb in the top layers of soil varies widely due to deposition and accumulation of atmospheric particulates from anthropogenic sources. The concentration of soil Pb generally decreases as distance from contaminating sources increases. The estimated Pb levels in the upper layer of soil beside roadways are typically 30–2,000 μg/g higher than natural levels, although these levels drop exponentially up to 25 m from the roadway (EPA 1986a). Soil adjacent to a smelter in Missouri had Pb levels in excess of 60,000 μg/g (Palmer and Kucera 1980). Soils adjacent to houses with exterior Pb-based paints may have Pb levels >10,000 μg/g (EPA 1986a). As a result of Pb reactions with the soil, extractable Pb in surface soil samples (0–5 cm depth) from an agricultural area near a car battery manufacturing plant (taken at 0.3 km from the source) decreased from 117 to 1 μg/g within 1 year after the plant stopped operating (Schalscha et al. 1987). Soil collected by scraping the top 2.5 cm of soil surface near homes and streetside in Louisiana and Minnesota contained median Pb concentrations of >840 μg/g in New Orleans and 265 μg/g in Minneapolis. In contrast, the small towns of Natchitoches, Louisiana, and Rochester, Minnesota had soil Pb concentrations of <50 and 58 μg/g, respectively. These data suggest that Pb-contaminated soil is a major source of Pb exposure in urban areas (Mielke 1993). As would be expected, soils in elementary school properties were also found to have the same pattern of Pb levels as the soils in the surrounding residences. Pb concentrations in soils collected from inner-city schools in New Orleans were higher (median concentration of 96.5 μg/g) than soils collected from mid-city (30.0 μg/g) and outer-city (16.4 μg/g) elementary schools (Higgs et al. 1999).

The former use of Pb in paints, particularly in older structures, is also a source of Pb in soil and within homes. Meilke and Gonzales (2008) reported median Pb concentrations of 76,603 mg/kg (464–317,151 mg/kg) and 416 mg/kg (24–63,313 mg/kg) for exterior and interior paints, respectively, in 40 paint chip samples collected from homes in metropolitan New Orleans. The authors noted that the age of the house is often used as a surrogate for the amount of Pb in paints; the mid-1920s being the peak use of leaded paint with declines until 1978. Demolition and renovation of buildings where leaded paint was used can result in transport of Pb to soil surrounding the building as well as indoor dust that contains Pb.

Pb concentrations were measured in residential transects through Lubbock, Texas. Pb concentrations through the city showed a trend of decreasing Pb concentrations with increasing distance from the city center, which also paralleled a decrease in the property age. The highest Pb concentration in the city center were 90.0–174.0 mg/kg, with a median of 35.4 mg/kg, and decreased out to the farther part of the residential transect to 6.0–9.0 mg/kg. The highest concentrations outside city development were 4.9 mg/kg (Brown et al. 2008).

Studies conducted in Maryland and Minnesota indicate that within large, light-industrial, urban settings such as Baltimore, the highest soil Pb levels generally occur near inner-city areas, especially where high traffic flows have long prevailed (Mielke et al. 1983, 1984, 1989) and that the amount of Pb in the soil is correlated with the size of the city (Mielke 1991). In 1981, soil Pb levels in the Minneapolis/St. Paul inner-city area were 60 times higher (423 μg/g) than levels found in rural Minnesota (6.7 μg/g), with almost all the increase (95%) resulting from the combustion of leaded gasoline. A study conducted in Minneapolis, Minnesota, after the Pb content of gasoline had been significantly reduced, found that median soil Pb levels taken from the foundations of homes, in yards, and adjacent to the street were 700, 210, and 160 μg/g, respectively; median soil Pb concentrations in comparable samples from the smaller city of Rochester, Minnesota, did not exceed 100 μg/g at any location tested (Mielke et al. 1989). The Minneapolis data suggested that average Pb levels were elevated in soil samples taken from the foundations of homes, but that Pb levels were low (<50 μg/g) in areas where children could be expected to play, such as parks that were located away from traffic, but were higher in play areas around private residences. Soil samples taken from around the foundations of homes with painted exteriors had the highest Pb levels (mean concentrations of 522 μg/g), but levels around homes composed of brick or stucco were significantly lower (mean concentration 158 μg/g) (Schmitt et al. 1988). Severely contaminated soils (levels as high as 20,136 μg/g) were located near house foundations adjacent to private dwellings with exterior Pb-based paint. Elevated soil Pb concentrations were found in larger urban areas, with 27, 26, 32, and 42% of the soil samples exceeding 300 μg/g Pb in Duluth, inner-city North Minneapolis, inner-city St. Paul, and inner-city South Minneapolis, respectively. Only 5% of the soil samples taken from the smaller urban areas of Rochester and St. Cloud, Minnesota, had Pb levels >150 μg/g. It has been suggested that the higher Pb levels associated with soils taken from around painted homes in the inner city are the result of greater atmospheric Pb content, resulting from the burning of leaded gasoline in cars and the washdown of building surfaces to which the small Pb particles adhere by rain (Mielke et al. 1989). A state-wide Minnesota study concluded that exterior Pb-based paint was the major source of contamination in severely contaminated soils located near the foundations of private residences and that aerosol Pb accounted for virtually all of the contamination found in soils removed

from the influence of Pb-based paint. Contamination due to Pb-based paint was found to be "highly concentrated over a limited area, while contamination due to aerosol Pb was found to be less concentrated, but more widespread" (Schmitt et al. 1988).

Pb was analyzed in dust wipes and soil samples from 67 public housing projects containing 487 dwelling units across the United States (Succop et al. 2001). A total of 5,906 dust wipes and 1,222 soil samples were included in the data set. The median soil levels were 194 ppm near the foundation, 177 ppm near the walkways, and 145 ppm elsewhere in the yard. The maximum level, 3,900 ppm, was found in a foundation sample. Median dust Pb loading from kitchens, living rooms, and two children's bedrooms were 151, 936, and 8,560 μg m^{-2} for floor window sills and window troughs, respectively. Thirteen percent of the floor samples and 30% of the window sill samples from the rooms exceeded the HUD Interim Dust Lead Standards of 431 and 2,690 μg m^{-2} for floor and window sill samples, respectively.

5.5.4 Paint

Weathering and deterioration of Pb-based paint can contribute to the Pb content of dust and soil (Clark et al. 2004; Hunt et al. 1992; Jaeger et al. 1998; Lucas et al. 2013; Marcus and Elias 1995 <in Beard and Iske 1995>; MPCA 1987). A state-wide Minnesota study concluded that exterior Pb-based paint was the major source of contamination in severely contaminated soils located near the foundations of private residences (MPCA 1987). A soil Pb study in Minneapolis, Minnesota, found that soil samples taken from around the foundations of homes with painted exteriors had a mean concentration of 522 μg/g, while soil samples taken from around the foundations of brick or stucco had a mean concentration of 158 μg/g (Schmitt et al. 1988). Pb-based paint, removed from surfaces by burning (gas torch or hot air gun), scraping, or sanding have been found to result, at least temporarily, in higher levels of exposure for families residing in these homes. A 2006 survey of U.S. housing stock estimated that 35% of 106 million housing units contained Pb-based paint and that approximately 20% of houses contained deteriorating Pb-based pant (HUD 2011).

5.5.5 Other Media

Pb has been detected in a variety of foods. Pb may be introduced into food through uptake from soil into plants or atmospheric deposition onto plant surfaces, during transport to market, processing, and kitchen preparation (EPA 1986a). The ban on leaded gasoline as well as the use of welded (non-soldered) food cans during the 1980s are largely responsible for the decreases in levels of Pb in the U.S. diet beginning in the 1980s (FDA 2006). The FDA analyzed samples of foods commonly eaten by toddlers and infants

for Pb and noted that levels of Pb in infant and toddler foods, on average, are relatively low (FDA 2016a). These results are summarized in Table 5-14. Selected data from the 2006–2011 FDA Total Diet Study Market Baskets are presented in Table 5-15 (FDA 2016b). Mean Pb levels in dairy products (e.g., milk, cheese, ice cream, cream, yogurt) were generally low or below the detection limit. The dairy product category with the highest Pb level was for low-fat fruit-flavored yogurt, with a mean concentration of 0.002 mg/kg for 24 analyses. Mean concentrations of Pb in fruits and vegetable were also generally low, with the highest concentrations in raisins (0.005 mg/kg), spinach (0.004 mg/kg), and lettuce (0.004 mg/kg). Mean concentration of Pb in baby foods ranged from not detected to 0.013 mg/kg. The highest levels reported were found in sweet potatoes (0.013 mg/kg), arrowroot cookies (0.012 mg/kg), grape juice (0.011 mg/kg), teething biscuits (0.008 mg/kg), and apple-cherry juice (0.008 mg/kg).

Table 5-14. Lead Levels in Foods Commonly Eaten by Toddlers and Infants

Product category	Average[a] (range) (µg/kg)	Number of samples
Cereal, infant/toddler (rice)	15.6 (5.0–82.0)	76
Cereal, infant/toddler (multigrain)	7.2 (6.0–8.0)	6
Cereal, infant/toddler (non-rice)	4.8 (0.4–17.0)	30
Apples[b]	3.3	10
Cereal, oat ring	7.8 (3.3–16.4)	30
Grapes	3.7 (3.3–7.6)	10
Juice, grape	5.6 (0.3–41.3)	30
Juice boxes and pouches	3.3 (0.3–17.0)	40
Peanut butter	5.3 (3.3–45.2)	29
Quinoa	22.2 (0.4–98.0)	30
Raisins	18.1 (1.8–151)	23
Stage 2 toddler foods	5.2 (1.0–22.2)	35
Teething biscuits	12.0 (2.0–131)	27
Toddler puffs	19.1 (3.3-–91.0)	31

[a]The average concentration reported for each product category was calculated using all values. For those samples with results below the detection limit, half of the detection limit was used to calculate the average.
[b]All of the apple samples were below the limit of detection.

Source: FDA 2016a

Table 5-15. Selected Mean Lead Concentrations in Food from the FDA Total Diet Study

Food	Mean (range) (mg/kg)[a]	Number of analyses	Number <LOD	LOD (mg/kg)
Syrup, chocolate	0.016 (0–0.027)	24	1	0.007
Apricots, canned in heavy/light syrup	0.015 (0–0.036)	24	1	0.007
Baby food, sweet potatoes	0.013 (0–0.034)	24	5	0.007
Peach, canned in light/medium syrup	0.013 (0–0.038)	24	2	0.007
Candy bar, milk chocolate, plain	0.013 (0–0.027)	24	5	0.01
Baby food, arrowroot cookies	0.012 (0–0.031)	24	9	0.01
Sweet potatoes, canned	0.012 (0–0.018)	24	2	0.007
Shrimp, boiled	0.012 (0–0.18)	24	18	0.01
Baby food, juice, grape	0.011 (0–0.02)	24	1	0.004
Fruit cocktail, canned in light syrup	0.011 (0–0.025)	24	4	0.007
Brownie	0.01 (0–0.032)	24	5	0.007

[a]Note: 1 mg/kg = 1,000 µg/kg.

FDA = U.S. Food and Drug Administration; LOD = limit of detection

Source: FDA 2016b

The U.S. Fish and Wildlife Service reported the concentrations of metals in a total of 315 composite samples of whole fish sampled from 109 stations nationwide from late 1994 to early 1995. For Pb, the geometric mean, maximum, and 85th percentile concentrations (µg/g wet weight) were 0.11, 4.88, and 0.22, respectively. The mean concentration of Pb was significantly lower than in the 1980–1981 survey. Pb concentrations in fish have declined steadily from 1976 to 1984, suggesting that reductions of leaded gasoline and controls on mining and industrial discharges have reduced Pb in the aquatic environment (Schmitt and Brumbaugh 1990).

In order to reduce Pb exposure from consumption of Pb-contaminated fish and shellfish, consumption advisories are issued by states recommending that individuals restrict their consumption of specific fish and shellfish species from certain water bodies where Pb concentrations in fish and shellfish tissues exceed the human health level of concern. This level of concern is set by individual state agencies and used to issue advisories recommending no consumption, or restricted consumption, of contaminated fish and shellfish from certain waterbody types (e.g., lakes and/or rivers). In 1995, the EPA Office of Water issued guidance to states on sampling and analysis procedures to use in assessing the health risks from consuming locally caught fish and shellfish. The risk assessment method proposed by EPA was specifically designed to assist states in developing fish consumption advisories for recreational and

subsistence fishers (EPA 1995a). These two groups within the general population consume larger quantities of fish and shellfish than the general population and frequently fish the same water bodies routinely. Because of this, these populations are at greater risk of exposure to Pb and other chemical contaminants if the waters they fish are contaminated. In 2007, eight advisories restricting the consumption of Pb-contaminated fish and shellfish were in effect in five states (Hawaii, Idaho, Washington, Kansas, and Missouri) and one territory (American Samoa) (EPA 2007b).

Elevated levels of Pb in the blood of cattle grazing near a Pb smelter have been reported, although no implications regarding Pb in beef were made. The mean Pb levels for the herd were highest near the smelter and decreased with distance. Ingestion of soil along with the forage was thought to be a large source of additional metal (Neuman and Dollhopf 1992). Evidence has also been shown for transfer of Pb to milk and edible tissue in cattle poisoned by licking the remains of storage batteries burned and left in a pasture (Oskarsson et al. 1992). Levels of Pb in muscle of acutely sick cows that were slaughtered ranged from 0.23 to 0.5 mg/kg (wet weight basis). Normal Pb levels in bovine meat from Swedish farms are <0.005 mg/kg. For eight cows that were less exposed, levels of Pb in milk taken 2 weeks after the exposure were 0.08±0.04 mg/kg. The highest Pb level found in the milk of eight cows studied for 18 weeks was 0.22 mg/kg. Pb in most milk samples decreased to values <0.03 mg/kg 6 weeks after exposure. Two affected cows delivered a calf at 35 and 38 weeks after the exposure. There was a high Pb level in the blood of the cows at the time of delivery, which suggests mobilization of Pb in connection with the latter stages of gestation and delivery. Pb levels in colostrum were increased as compared to mature milk samples taken 18 weeks after exposure. The concentration of Pb in milk produced after delivery decreased rapidly with time and was almost down to the limit of detection in mature milk.

In a survey, 324 multivitamin-mineral products were analyzed for Pb content (Mindak et al. 2008). Estimates of Pb exposure from these products were derived for four groups summarized in Table 5-16. The overall median value for Pb exposure was 0.576 µg/day. Five samples would have provided exposures that exceeded 4 µg/day. The authors reported that the estimates of Pb exposures were below the provisional total tolerable intake levels for the four population groups (Mindak et al. 2008). Twenty-one elements, including Pb, were analyzed in various botanical and dietary supplements; Pb concentrations ranged from not detected to 4.21 µg/g. None of the products analyzed would result in a maximum exposure that exceeds a tolerable level of exposure (Avula et al. 2010).

Table 5-16. Estimated Median and Maximum Lead Exposures

Population group	Median (µg/day)	Maximum (µg/day)
Young children (0–6 years)	0.123	2.88
Older children (7+ years)	0.356	1.78
Pregnant or lactating women	0.845	8.97
Adult women	0.842	4.92

Source: Adapted with permission from Mindak et al. (2008), American Chemical Society.

Many non-Western folk remedies used to treat diarrhea or other ailments may contain substantial amounts of Pb. Examples of these include: Alarcon, Ghasard, Alkohl, Greta, Azarcon, Liga, Bali Goli, Pay-loo-ah, Coral, and Rueda. In addition, an adult case of Pb poisoning was attributed to an Asian remedy for menstrual cramps known as Koo Sar. The pills contained Pb at levels as high as 12 ppm (CDC 1998). The source of the Pb was thought to be in the red dye used to color the pills. Pb was the most common heavy metal contaminant/adulterant found in samples (n=54) of Asian traditional remedies available at health food stores and Asian groceries in Florida, New York, and New Jersey (Garvey et al. 2001). Sixty percent of the remedies tested would give a daily dose of Pb in excess of 300 mg when taken according to labeling instructions. Pb poisoning has been caused by ingestion of a Chinese herbal medicine to which metallic Pb was added to increase its weight and sales price (Wu et al. 1996). Ayurveda is a traditional form of medicine practiced in India and other South Asian countries; the medications used often contain herbs, minerals, metals, or animal products and are made in standardized and nonstandardized formulations (CDC 2004). CDC (1998, 2002b) reported cases of elevated PbBs in children after consuming candy from Mexico or using various folk remedies. Elevated PbBs were reported in two 7-year-old children in Rhode Island. A sample of litargirio, which was used as an antiperspirant/deodorant, found in the home contained 79% Pb (CDC 2005).

During 2011–2012, six cases of Pb poisoning were associated with the use of 10 oral Ayurvedic medications made in India. Pb concentrations in these medications were as high as 2.4%. Blood Pb levels of these women ranged from 16 to 64 µg/dL (CDC 2012c). In 2004–2012, the New York City Department of Health and Mental Hygiene identified 22 oral medications, supplements, or remedies containing high levels of heavy metals, including Pb (Table 5-17).

Table 5-17. Lead Content in Ayurvedic Medications and Other Health Remedies

Product	Country where manufactured	Country where purchased	Lead content (ppm)
Calabash Chalk (Nzu)	Unknown	United States	6.6
Emperor's Tea Pill (concentrated)	China	United States	5,400
Garbha Chintamani Ras (Vrihat) (Swarna Yukt)	India	India	120
Garbha Dharak Yog	India	India	110
Garbhapal Ras	India	India	22,000
Garbhapal Ras	India	United States	15,000
Hepatico Extract (concentrated)	China	United States	5,900
Jambrulin	India	United States	243,000
Kankayan Bati (Gulma)	India	United States	12
Lakshmivilash Ras (Nardiya)	India	United States	260
Laxmana Louh	India	India	180
Maha Sudarshan	India	United States	41
Mahashakti Rasayan	India	India	9,400
Mahayogaraj Guggulu (enriched with silver)	India	United States	47,000
Ovarin	India	India	24,000
Pigmento	India	India	7.3
Pregnita	India	India	12,000
Sorin	India	India	46,707
Tierra Santa	Mexico	United States	13
Vasant Kusumakar Ras (with Gold and Pearl)	India	India	29
Vatvidhwansan Ras	India	United States	20,000
Vita Breath	United States	United States	1,100

Source: CDC 2012c

A study was conducted in an urban neighborhood in Chicago in order to gauge the levels of Pb in an array of fruits, vegetables, and herbs (Finster et al. 2004). The soil Pb concentrations where the plants were sampled varied from 27 to 4,580 ppm (median 800 ppm, geometric mean 639 ppm). Detectable Pb levels in the edible fruit, vegetables, and herbs sampled ranged from 11 to 81 ppm. Only one fruiting vegetable (cucumber 81 ppm) among the 52 sampled had detectable levels of Pb in the edible portion. However, 12 of the 31 leafy vegetables and herbs sampled contained Pb in the edible shoot part of the plant (range, 11–60 ppm). The Pb concentrations in the four samples of root vegetables ranged from 10 to 21 ppm. No significant correlation was found between the Pb concentrations in the edible portion of plant and the soil Pb level.

Pb may leach from Pb crystal decanters and glasses into the liquids they contain. Port wine that contained an initial concentration of 89 µg/L Pb was stored for 4 months in crystal decanters containing up to 32% Pb oxide. At the end of 4 months, Pb concentrations in the port were 5,331, 3,061, and 2,162 µg/L in decanters containing 32, 32, and 24% Pb oxide, respectively. Pb was also found to elute from Pb crystal wine glasses within minutes. Mean Pb concentrations in wine contained in 12 glasses rose from 33 µg/L initially to 68, 81, 92, and 99 µg/L after 1, 2, 3, and 4 hours, respectively (Graziano and Blum 1991).

Hair dyes and some cosmetics may contain Pb compounds (Cohen and Roe 1991). Hair dyes formulated with Pb acetate may have Pb concentrations 3–10 times the allowable concentration in paint. Measured Pb concentrations of 2,300–6,000 µg of Pb/gram of product have been reported (Mielke et al. 1997). Pb acetate is soluble in water and easily transferred to hands and other surfaces during and following application of a hair dye product. Measurements of 150–700 µg of Pb on each hand following application have been reported (Mielke et al. 1997). In addition to transfer of Pb to the hand-to-mouth pathway of the person applying the product, Pb can be transferred to any other surface (comb, hair dryer, outside of product container, counter top, etc.) that comes into contact with the product. It is also on the hair that it is applied to and the hands applying it. Objects coming into contact with hair dyed with a Pb-containing product also become contaminated. A dry hand passed through dry hair dyed with a Pb-containing product in cream form was been shown to pick up about 786 µg of Pb. A dry hand passed through dry hair dyed using foam or liquid Pb-containing hair dye products picked up less Pb: 69 µg/hand for foam products and 73 µg/hand for liquid products (Mielke et al. 1997). An elevated PbB (12 µg/dL) in an infant was observed after the use of tiro, a Nigerian eye cosmetic applied to the infant's eyes (CDC 2012a). Elevated PbBs (27.0 and 33.5 µg/dL) were reported in two young children in New Mexico after the use of kajal, a cosmetic imported from Afghanistan, that was applied to the children's eyelids. The kajal was reported to contain 54% Pb (CDC 2013).

Cases of Pb poisoning have been related to less common sources of exposure. Illicit "moonshine" whiskey made in stills composed of Pb-soldered parts (e.g., truck radiators) may contain high levels of Pb. Detectable levels of Pb with a maximum concentration of 5.3 mg/L were found in 7 of 12 samples of Georgia moonshine whiskey (Gerhardt et al. 1980). Of the 115 suspected moonshine samples seized by local law enforcement between 1995 and 2001 and analyzed by the Bureau of Alcohol, Tobacco, and Firearms, 33 samples (28.7%) contained Pb levels >300 µg/dL. The median and maximum levels were 44.0 and 53,200 µg/dL, respectively (Parramore et al. 2001).

Firing of Pb ammunition may result in exposure to Pb aerosols and dusts generated during gun or rifle discharge at levels up to 1,000 μg/m^3 (EPA 1985c), from Pb pellets ingested by or imbedded in animals that are used as food sources, and from Pb pellets or fragments imbedded in humans from shooting incidents (see Appendix C, Ingestion of Lead Debris). Exposures to airborne Pb dust from firearm discharge in indoor shooting ranges has been shown to result in increases in PbBs that are 1.5–2 times higher than preexposure concentrations (Greenberg and Hamilton 1999; Gulson et al. 2002). However, the use of copper-jacketed bullets, nonlead primers, and well-ventilated indoor firing ranges lessen the impact of airborne Pb on blood Pb levels (Gulson et al. 2002).

A Pb poisoning hazard for young children exists in imported vinyl miniblinds that had Pb added to stabilize the plastic. Over time, the plastic deteriorates to produce Pb dust that can be ingested when the blinds are touched by children, who then put their hands in their mouths (CPSC 1996). The U.S. Consumer Product Safety Commission (CPSC) has requested that manufacturers change the manufacturing process to eliminate the Pb. As a consequence, vinyl miniblinds should now be Pb-free. The CPSC recommends that consumers with young children remove old vinyl miniblinds from their homes and replace them with new miniblinds made without added Pb or with alternative window coverings.

Inexpensive metallic jewelry items specifically intended for children and teenagers have been shown to contain varying levels of Pb (Maas et al. 2005). A total of 311 chemical assays conducted using 285 jewelry items purchased in 20 different stores in California revealed that a considerable amount of Pb was added to the items, presumably to increase their weight or to impart some type of metallic coating to the surface of the item. The mean weight percentage of Pb for all 311 assays was 30.6%. Of the 311 samples tested, 169 contained at least 3% Pb by weight in at least one portion of the jewelry piece and 123 of the samples were found to contain >50% Pb by weight (Maas et al. 2005). In addition, 62 pieces of the purchased jewelry were tested for surface levels of Pb that could potentially be transferred dermally through the routine handling of these pieces. Using standard laboratory wipes, the surface of the jewelry pieces were wiped for a total of 20 seconds and subsequently analyzed for Pb content. Mean Pb levels in the wipes ranged from 0.06 to 541.97 μg. The authors characterized the potential Pb exposure from these dermal transfer experiments as either low exposure (<1 μg of Pb transferred to the laboratory wipe), moderate exposure (1–10 μg of Pb transferred to the laboratory wipe), high exposure (10–50 μg of Pb transferred to the laboratory wipe), and very high exposure (>50 μg of Pb transferred to the laboratory wipe). Approximately 35% of the 62 pieces tested were characterized as

having low exposure, 48% were characterized as moderate exposure, 11% were characterized as high exposure, and 5% were characterized as very high exposure (Maas et al. 2005).

5.6 GENERAL POPULATION EXPOSURE

Measurements of Pb in blood, urine, and tissues (postmortem) have been used to assess exposures of individuals to Pb. Table 5-18 shows the lowest limit of detections that are achieved by analytical analysis of blood, urine and tissues.

Table 5-18. Lowest Limit of Detection Based on Standards[a]

Media	Detection limit	Reference
Whole blood/urine/tissue	0.05 µg Pb/g blood or mL urine	NIOSH 1994d, Method 8003
	1 µg/100 g blood; 0.2 µg/g tissue	NIOSH 1994a, Method 8005
Animal tissue	0.1 µg/g (ICP-MS or GFAA)	NOAA 1998

[a]Detection limits based on using appropriate preparation and analytics. These limits may not be possible in all situations.

GFAA = graphite furnace atomic absorption; ICP-MS = inductively coupled plasma-mass spectrometry

Prior to the 1980s, aerolized Pb emissions from the use of leaded gasoline was the main source of Pb exposure for the general U.S. population. Aerolized Pb can be either inhaled or ingested after deposition on surfaces and food crops. Adult Pb exposures tend to be limited to occupational or recreational sources. For children, the primary source of Pb exposure is from surface dusts (on the ground or entrained) that contain Pb from a variety of sources including deteriorated Pb-based paint (Bornschein et al. 1986; CDC 2009; Dixon et al. 2009; Egeghy et al. 2005; EPA 1996c; Garavan et al. 2008; Gulson et al. 2009; Lanphear and Roghmann 1997; Lanphear et al. 1998a; Lewin et al. 1999; Malcoe et al. 2002; Mielke et al. 2007; Succop et al. 1998; Von Lindern et al. 2003, 2016; Zahran et al. 2013). Young children are particularly vulnerable to Pb exposure because of hand-to-mouth activity, which contributes to ingestion of Pb in surface dusts. Pb in the fine particle fraction of surface dusts (<150 µm) readily adheres to the skin surface, from which it can be inadvertently ingested from hand-to-mouth activity (Choate et al. 2006a, 2006b; Clausing et al. 1987; Davis and Mirick 2006; Davis et al. 1990; Siciliano et al. 2009; Yamamoto et al. 2006). Several studies have attempted to quantify soil and dust ingestion in children (Chien et al. 2017; Ozkaynak et al. 2011; Sedman et al. 1994; Stanek et al. 2012; Von Lindern et al. 2016; Wilson et al. 2013) and adults (Calabrese et al. 1990; Doyle et al. 2012; Irvine et al. 2014; Stanek et al. 1997).

Although air Pb can be a direct pathway of exposure in children, it can also be an indirect pathway from its effect on Pb concentration in surface dusts (Brunekreef 1984; Hayes et al. 1994; Hilts 2003; Rabinowitz et al. 1985; Schnaas et al. 2004; Schwartz and Pitcher 1989; Tripanthi et al. 2001). Second-hand smoke may also contribute to increased Pb exposure (Apostolou et al. 2012; Mannino et al. 2003; Richter et al. 2013). Dietary sources of Pb can originate from direct or indirect transfer of atmospheric Pb emissions to secondary media such as water, food crops, game, and fish. Pb in the maternal system can also be transferred to the fetus during gestation and to the nursing infant (EPA 2014c).

Several studies provided data with which dietary intakes of Pb for the general population in the United States have been estimated (FDA 2016 <TDS 2006-2011>; FDA 2016 -< Combination Metals Testing>; FDA 2016 <TDS 2006-2011>). An analysis of individual food intakes and PbB from NHANES (2006–2008) estimated that diet explained approximately 2.9% of the variations of PbB in children and 1.6% in adults (Davis et al. 2014). A randomized survey of 250 individuals (adults and children) from the Midwest United States conducted over the period 1995–1997 estimated average dietary Pb intake to be approximately 10 μg/day (Clayton et al. 1999). The EPA has estimated mean dietary Pb intakes in children ages 6–84 months to be approximately 2 μg/day (EPA 2014c). The ban on the use of welded (non-soldered) food cans during the 1980s has resulted in a decrease in Pb exposure from foods (FDA 2006). In recent surveys, the mean Pb levels in dairy products (e.g., milk, cheese, ice cream, cream, yogurt) were generally low or below the detection limit. Mean concentrations of Pb in fruits and vegetable were also generally low. Mean concentration of Pb in baby foods ranged from not detected to 0.013 mg/kg. Possible sources of Pb in food samples include introduction during processing or preparation with drinking water contaminated with Pb, deposition of Pb onto raw materials for each food, and Pb exposure in livestock that produce dairy or meat ingredients (EPA 2014c). Pb has also been reported in home-prepared reconstituted infant formula. Although, at one time, use of Pb solder in formula containers contributed to PbB from formula consumption (Ryu et al. 1983), this practice was phased out after 1970 in the United States and subsequently banned (FDA 1995). However, tap water remains a potential source of Pb in home-prepared formula at locations where tap water Pb concentrations are elevated. In a study conducted in the Boston area in 1997, 2 of 40 samples of home-prepared formula had Pb concentrations >15 μg/L. In both cases, the reconstituted formula had been prepared using cold tap water run for 5–30 seconds, drawn from the plumbing of houses >20 years old. Pb-containing ceramic ware used in food preparation has also been associated with childhood Pb exposure in children of Hispanic ethnicity in San Diego County, California. One study (Gersberg et al. 1997) used the IEUBK

Model to determine that dietary Pb exposure from beans prepared in Mexican ceramic bean pots may account for a major fraction of blood Pb burden in children whose families use such ceramic ware.

Generally, Pb is unlikely to be found in source water used for drinking water unless there is a specific source of contamination. While Pb was restricted in plumbing materials in 1986, older homes and neighborhoods may still contain Pb service lines, Pb connections, Pb solder, or other Pb-based plumbing materials that may contaminate drinking water during its delivery from its source to homes. Corrosion of these older plumbing materials can result in leaching of Pb into drinking water (CDC 2012b; Hanna-Attisha et al. 2016). Flint, Michigan is an example of how an aging water system can result in Pb contamination in drinking water. The original water source in Flint, Michigan was characterized as having very low corrosivity for Pb (i.e., low chloride and low chloride-to-sulfur mass ratio) and was treated with an orthophosphate corrosion inhibitor. While the delivery system contained Pb, its surface was covered in a protective layer of corrosion materials and the Pb was unavailable to leach into the drinking water. When the water source was changed to the Flint River in 2014, the chemistry of the water was very different (i.e., high chloride, high chloride-to-sulfur mass ratio), favoring disruption of the protective corrosion layer, which allowed the Pb to leach into the drinking water. In addition, the source water was not treated with a corrosion inhibitor that could help to maintain the protective layer in the system (Hanna-Attisha et al. 2016). Pb concentration in first-draw tap water tends to be higher than after the plumbing system has been flushed. Gulson et al. (1997a) measured Pb in household water throughout the day when the plumbing system of an unoccupied test house was not flushed. Water concentration data ranged from 119 μg/L for the initial (first-draw) sample to 35–52 μg/L for hourly samples to 1.7 μg/L for a fully flushed sample.

Other less common sources of Pb exposure also exist. Exposure may also result from engaging in hobbies that use Pb (e.g., leaded solder is used in making stained glass, molten Pb used in casting, leaded glazes and frits are used in making pottery, and Pb compounds as coloring agents in glassblowing) (Grabo 1997). The use of inadequately glazed or heavily worn earthenware vessels for food storage and cooking may result in Pb exposure (CDC 1985; EPA 1986a). Various folk remedies and Ayurvedic medication (CDC 1998, 2004, 2012c; Garvey et al. 2001; Wu et al. 1996) and some cosmetics (Mielke et al. 1997) may also be sources of Pb exposure. Moonshine consumption was strongly associated with elevated PbBs (Morgan and Parramore 2001). A 2000 study found a median PbB of 11 μg/dL among 35 moonshine consumers versus 2.5 μg/dL in 68 randomly-selected nonmoonshine consumers (Parramore et al. 2001). Exposure to infants and children can occur from mouthing of leaded jewelry and toys containing Pb or painted with leaded paint (CDC 2018c).

Plastic food wrappers may be printed with pigments that contain Pb chromates. Plastic wrappers used for 14 different national brands of bread collected in New Jersey contained a mean concentration of 26 mg of Pb for a bag size of 2,000 cm^2. A survey of 106 homemakers who buy such breads indicated that 39% of them reused the bags and 16% of the respondents turned the bags inside out to reuse them, suggesting that the potential exists for Pb leaching from the paint into the stored food (Weisel et al. 1991).

Blood Pb levels measured as a part of the NHANES revealed that between 1976 and 1991, the mean PbBs of the U.S. population aged 1–74 years old dropped 78%, from 12.8 to 2.8 µg/dL. The prevalence of PbBs ≥10 µg/dL also decreased sharply from 77.8 to 4.3%. The major cause of the observed decline in PbBs is most likely the removal of 99.8% of Pb from gasoline and the removal of Pb from soldered cans (Pirkle et al. 1994). Data from the Fourth National Report on Human Exposure to Environmental Chemical are summarized in Tables Table 5-19 and Table 5-20, which provide geometric means of Pb levels in the blood and urine in segments of the U.S. population.

Table 5-19. Geometric Mean Blood Lead Levels (µg/dL) and the 95th Percentile Confidence Interval, by Race/Ethnicity, Sex, and Age

	Survey years	Geometric mean (95% confidence interval)	Sample size
Total	11–12	0.973 (0.916–1.04)	7,920
	13–14	0.858 (0.813–0.906)	5,215
	15–16	0.820 (0.772–0.872)	4,988
Age group			
1–5 years	11–12	0.970 (0.877–1.07)	713
	13–14	0.782 (0.705–0.869)	818
	15–16	0.758 (0.675–0.850	790
6–11 years	11–12	0.681 (0.623–0.744)	1,048
	13–14	0.567 (0.529–0.607)	1,075
	15–16	0.571 (0.523–0.623)	565
12–19 years	11–12	0.554 (0.511–0.601)	1,129
	13–14	0.506 (0.464–0.551)	627
	15–16	0.467 (0.433–0.504	1,023
20 years and older	11–12	1.09 (1.03–1.16)	5,030
	13–14	0.967 (0.921–1.02)	2,695
	15–16	0.920 (0.862–0.982)	2,610

Table 5-19. Geometric Mean Blood Lead Levels (µg/dL) and the 95th Percentile Confidence Interval, by Race/Ethnicity, Sex, and Age

	Survey years	Geometric mean (95% confidence interval)	Sample size
Gender			
Males	11–12	1.13 (1.06–1.21)	3,968
	13–14	0.994 (0.919–1.08)	2,587
	15–16	1.13 (1.06–1.21)	3,968
Females	11–12	0.842 (0.796–0.890)	3,952
	13–14	0.746 (0.715–0.777)	2,628
	15–16	0.735 (0.679–0.795)	2,500
Race/ethnicity			
Mexican Americans	11–12	0.838 (0.767–0.916)	1,077
	13–14	0.746 (0.685–0.813)	969
	15–16	0.704 (0.659–0.759)	994
Non-Hispanic blacks	11–12	0.998 (0.947–1.05)	2,195
	13–14	0.871 (0.787–0.963)	1,119
	15–16	0.856 (0.763–0.962	1,070
Non-Hispanic whites	11–12	0.993 (0.914–1.08)	2,493
	13–14	0.882 (0.820–0.950)	1,848
	15–16	0.835 (0.774–0.900)	1,511
All Hispanics	11–12	0.855 (0.793–0.922)	1,931
	13–14	0.742 (0.695–0.793)	1,481
	15–16	0.703 (0.658–0.750)	1,664
Asians	11–12	1.15 (1.06–1.24)	1,005
	13–14	1.01 (0.923–1.11)	510
	15–16	1.07 (0.976–1.18)	479

Source: CDC 2018a

Table 5-20. Geometric Mean Urine Lead Levels (µg/dL) and the 95th Percentile Confidence Interval, by Race/Ethnicity, Sex, and Age

	Survey years	Geometric mean (95% confidence interval)	Sample size
Total	11–12	0.360 (0.328–0.396)	2,504
	13–14	0.277 (0.257–0.298)	2,664
Age group			
6–11 years	11–12	0.346 (0.292–0.410)	399
	13–14	0.222 (0.192–0.258)	402
12–19 years	11–12	0.259 (0.219–0.305)	390
	13–14	0.201 (0.166–0.245)	451
20 years and older	11–12	0.381 (0.348–0.416)	1,715
	13–14	0.297 (0.280–0.315)	1,811

Table 5-20. Geometric Mean Urine Lead Levels (µg/dL) and the 95th Percentile Confidence Interval, by Race/Ethnicity, Sex, and Age

	Survey years	Geometric mean (95% confidence interval)	Sample size
Gender			
Males	11–12	0.414 (0.367–0.466)	1,262
	13–14	0.315 (0.295–0.337)	1,318
Females	11–12	0.316 (0.282–0.355)	1,242
	13–14	0.245 (0.222–0.269)	1,346
Race/ethnicity			
Mexican Americans	11–12	0.372 (0.320–0.431)	317
	13–14	0.277 (0.240–0.319)	453
Non-Hispanic blacks	11–12	0.431 (0.385–0.483)	669
	13–14	0.371 (0.320–0.429)	581
Non-Hispanic whites	11–12	0.346 (0.311–0.385)	820
	13–14	0.267 (0.245–0.290)	985
All Hispanics	11–12	0.372 (0.327–0.423)	573
	13–14	0.270 (0.239–0.305)	701
Asians	11–12	0.383 (0.341–0.429)	353
	13–14	0.257 (0.230–0.287)	292

Source: CDC 2018a

The Adult Blood Lead Epidemiology and Surveillance (ABLES) program tracks cases of adult (aged ≥16 years) elevated PbBs from workplace exposure. In 2012, 41 states submitted data on 7,529 adults with PbBs ≥25 µg/dL and 38 states submitted data on 27,218 adults with PbBs ≥10 µg/dL (Alarcon 2015). PbBs ≥10 µg/dL declined from 26.6 adults per 100,000 employed in 2010 to 22.5 per 100,000 employed in 2012. In 2012, 53% of adults with PbBs ≥10 µg/dL were aged 40–64 years and 33.3% were aged 25–39 years; most (91.5%) were males. During 2002–2012, the annual proportion of PbBs ≥25 µg/dL from occupational exposures was 94.7% from participating states. Table 5-21 presents the number and percentage of adults (i.e., employed persons ≥16 years) with PbB ≥25 µg/dL by industry (NIOSH 2017).

Table 5-21. Adults with Blood Lead Concentration (PbB) ≥25 µg/dL by Industry

Industry	Number	Percent
Total, manufacturing industries	873	48.5
Storage battery manufacturing	340	38.9
Nonferrous metal (except copper and aluminum) rolling, drawing, extruding, and alloying	196	22.5

Table 5-21. Adults with Blood Lead Concentration (PbB) ≥25 µg/dL by Industry

Industry	Number	Percent
Alumina and aluminum production and processing	102	11.7
Nonferrous metal foundries	56	6.4
Other basic inorganic chemical manufacturing	40	4.6
All other fabricated metal product manufacturing	33	3.8
Other manufacturing industries	106	12.1
Total, construction industries	450	25.0
Highway, street, and bridge construction	131	29.1
Painting and wall covering contractors	97	21.6
Residential building construction	65	14.4
Plumbing, heating, and air-conditioning contractors	55	12.2
All other specialty trade contractors	25	5.6
Site preparation contractors	20	4.4
Other heavy and civil engineering construction	17	3.8
Other construction industries	40	8.9
Total, services (except public safety) industries	131	7.3
All other amusement and recreation industries	61	46.6
Automotive mechanical and electrical repair and maintenance	14	10.7
Remediation services	11	8.4
Other services (except public safety industries)	45	34.4
Total, mining industries	63	3.5
Copper, nickel, lead, and zinc mining	61	96.8
Support activities for mining	2	3.2
Total, other/missing industry information	284	15.8

Source: NIOSH 2017

Raymond and Brown (2015a, 2015b, 2017) and analyzed the 2007–2012 and 2009–2014 datasets from the Childhood Blood Lead Surveillance (CBLS) system. In 2007, a total of 38 states identified and reported 37,289 children (<6 years) with PbB ≥10 µg/dL. In 2012, a total of 30 jurisdictions identified and reported approximately 138,000 children (<6 years) with PbB ≥5 µg/dL. In 2012, federal funding ended and several states lost their state-wide Pb programs and in 2013, the number of states reporting data declined, as did the number of children reported to the CDC with PbB ≥5 µg/dL. In October 2013, federal funding resumed and in 2013, 27 states, the District of Columbia, and New York City reported data. In 2014, 30 states, the District of Columbia, and New York City reported data. Table 5-22 summarizes the number and rate per 100,000 children aged <5 years with blood Pb levels 5–9 µg/dL reported in the 2010–2014 CBLS system. PbBs >10 µg/dL continue to be more prevalent among children

with known risk factors, such as minority race or ethnicity, urban residence, residing in homes built prior to the 1950s, and low family income (CDC 2009).

Table 5-22. Number and Rate per 100,000 Children Aged <5 Years with Blood Lead Levels 5–9 µg/dL in the Childhood Blood Lead Surveillance System, United States, 2010–2014

	<1 Year		1–4 Years	
Year	Number	Rate	Number	Rate
2010[a]	18,598	448.48	137,887	805.62
2011[b]	13,981	352.69	130,838	810.56
2012[c]	7,876	199.74	95,854	596.58
2013[d]	5,494	138.26	57,293	360.46
2014[e]	5,904	148.51	70,680	444.49

[a]37 jurisdictions reporting.
[b]36 jurisdictions reporting.
[c]30 jurisdictions reporting.
[d]29 jurisdictions reporting.
[e]32 jurisdictions reporting.

Source: Raymond and Brown 2017

Various studies suggest that ingestion of game hunted with Pb shot is associated with increased PbBs. A 1992 survey of Pb in blood of 492 Inuit adults from the Arctic region of Quebec, Canada resulted in geometric mean Pb concentrations of 0.42 µmol/L (8.7 µg/dL), with a range of 0.04–2.28 µmol/L (0.8–47 µg/dL). Analysis of variance revealed that smoking, age, and consumption of waterfowl were associated with elevated Pb levels (Dewailly et al. 2001). Umbilical cord blood was collected from a cohort of Inuit newborns from northern Quebec, where the population consumes game killed with Pb shot; the geometric cord blood Pb concentration was 3.9 µg/dL (range 0.2–27 µg/dL). In this study, 7% of Inuit newborns had cord PbBs above 10 µg/dL as compared to 0.16% of the non-Inuit population in southern Quebec (Lévesque et al. 2003). Johansen et al. (2006) collected blood samples from 50 men in Nuuk, Greenland to study the relationship between the consumption of birds hunted with Pb shot and PbBs. Men who regularly ate hunted birds killed with Pb shot had mean PbB ranging from 6.2 µg/dL in the group eating 0.1–5 bird equivalents per month to 12.8 µg/dL in those eating >30 bird equivalents per month. In addition, levels were highest in mid-winter when consumption of hunted birds was highest. Those who did not consume hunted birds had a mean PbB of 1.5 µg/dL.

Second-hand smoke may also contribute to increased Pb exposure (Apostolou et al. 2012; Mannino et al. 2003; Richter et al. 2013). Pb is a component of tobacco and tobacco smoke, and smokers often have

higher Pb blood levels than nonsmokers (Bonanno et al. 2001; Mannino et al. 2003). Using data from the NHEXAS EPA Region V study, PbB levels in smokers and nonsmokers were analyzed and a correlation between tobacco smoke and exposure levels was observed (Bonanno et al. 2001). The mean PbBs in smokers, nonsmokers exposed to environmental tobacco smoke (ETS), and nonsmokers without ETS were 2.85, 2.06, and 1.81 µg/dL, respectively (Bonanno et al. 2001). Recent Pb urine concentrations for the U.S. adult population from the NHANES by smoking status are presented in Table 5-23.

Table 5-23. Geometric Mean Urine Lead Levels (µg/dL) and the 95th Percentile Confidence Interval by Smoking Status

	Survey years	Geometric mean (95% confidence interval)	Sample size
Cigarette smokers			
Total	11–12	2.36 (1.71–4.62)	876
	13–14	1.51 (1.30–1.91)	957
Age group			
20–49 years	11–12	1.78 (1.41–3.07)	522
18–49 years	13–14	1.34 (1.13–1.92)	583
50 years and older	11–12	3.35 (1.62–6.83)	354
	13–14	1.72 (1.40–2.03)	374
Gender			
Males	11–12	3.07 (1.73–5.03)	527
	13–14	1.91 (1.48–2.14)	512
Females	11–12	1.58 (1.14–3.45)	349
	13–14	1.30 (1.12–1.41)	445
Nonsmokers[a]			
Total	11–12	1.38 (1.25–1.58)	1,343
	13–14	1.16 (0.950–1.51)	1,487
Age group			
20–49 years	11–12	1.26 (1.02–1.38)	671
18–49 years	13–14	0.880 (0.720–1.04)	778
50 years and older	11–12	1.63 (1.29–2.16)	672
	13–14	1.48 (1.12–2.52)	709
Gender			
Males	11–12	1.61 (1.18–2.13)	635
	13–14	1.51 (1.04–2.68)	663
Females	11–12	1.32 (1.06-1.38)	708
	13–14	0.238 (0.219–0.258)	824

[a]Cigarette nonsmokers who used other tobacco products were excluded.

Source: CDC 2018b

Studies have been conducted to determine exposure of firearm instructors to Pb at outdoor firing ranges when either nonjacketed (pure Pb) or jacketed (copper-coated) bullets were used. Instructors are likely to have higher exposure than shooters because they spend more time at the range. In studies at an outdoor range in Virginia, the mean breathing zone Pb level when nonjacketed bullets were fired was 67.1 μg/m^3 for one instructor and 211.1 μg/m^3 for another (Tripathi and Llewellyn 1990). When jacketed bullets were used, breathing zone levels decreased to ≤8.7 μg/m^3. PbBs of the instructors did not exceed the OSHA lead standard's medical removal level of 2.4 μmol/L (60 μg/dL) in either case (OSHA 2016a). When shooters fired conventional Pb bullets, their mean exposures to airborne Pb were 128 μg/m^3 in the personal breathing zone and 68 μg/m^3 in the general area. When totally copper-jacketed Pb bullets were fired, the mean breathing zone and general area air sample concentrations were 9.53 and 5.80 μg/m^3, respectively (Tripathi and Llewellyn 1990). At an outdoor uncovered range in Los Angeles, instructors who spent an average of 15–20 hours/week behind the firing line were found to be exposed to breathing zone Pb concentrations of 460 and 510 μg/m^3 measured as 3-hour, time-weighted averages. The PbB of one instructor reached 3.38 μmol/L (70 μg/dL). After reassignment to other duties, repeat testing indicated his PbB had dropped to 1.35 μmol/L (28 μg/dL) (Goldberg et al. 1991).

In 1991, NIOSH conducted a survey of the Federal Bureau of Investigations (FBI) Firearms Training Unit firing ranges and related facilities to determine occupational Pb exposures among FBI and Drug Enforcement Agency (DEA) firing range personnel (NIOSH 1996b). Sixty-one personal breathing-zone and 30 area samples for airborne Pb were collected. Exposures ranged up to 51.7 μg/m^3 (mean, 12.4 μg/m^3), 2.7 μg/m^3 (mean, 0.6 μg/m^3), and 4.5 μg/m^3 (mean, 0.6 μg/m^3) for range instructors, technicians, and gunsmiths, respectively. Exposure of custodians ranged from nondetectable to 220 μg/m^3 during short-term cleaning of a large indoor range. Carpet dust sampling of dormitory rooms of students who practiced at the firing ranges revealed higher ($p<0.0005$) dust-Pb concentrations when compared to nonstudent dormitories (dust-Pb concentration range of 116–546 μg/g with a geometric mean of 214 μg/g in the student's rooms versus a dust-Pb concentration range of 50–188 μg/g with a geometric mean of 65 μg/g for the nonstudent rooms). This suggested that the students were contaminating their living quarters with Pb.

The American Academy of Pediatrics (AAP) (1998, 2005) concluded that although monitoring data demonstrate a decline in PbBs, Pb remains a common, preventable, environmental health threat. Most Pb poisoning in children is the result of dust and chips from deteriorating Pb paint on interior surfaces (AAP 2005, 2016; ATSDR 2017). The AAP supported the CDC guidelines endorsing universal screening in certain areas and targeted screening for children at high risk (CDC 1997b, 2005). Many children continue

to be at risk for ingestion of Pb-based paint and of soil and dust contaminated through the deterioration of Pb-based paint and the residues from combustion of leaded gasoline. A 1974 study indicated that elevated PbBs in children were most likely a result of ingesting Pb-contaminated soil, and that the most likely source was Pb-based paint rather than Pb from automotive exhaust (Ter Haar and Aronow 1974). However, more recent studies have shown that children with the highest PbBs live in areas with high traffic flow where Pb particles in the air may fall directly to the soil or adhere to the outer surfaces of building and wash to the soil with rain (Mielke et al. 1989, 2008, 2010). The CDC concluded that a common source of Pb exposure for children who have elevated PbB is Pb-based paint that has deteriorated into paint chips and Pb dusts (CDC 1997c, 2012d).

Pb can readily cross the placenta; therefore, exposure of women to Pb during pregnancy results in uptake by the fetus. Furthermore, since the physiological stress of pregnancy may result in mobilization of Pb from maternal bone, fetal uptake of Pb can occur from a mother who was exposed to Pb before pregnancy, even if no Pb exposure occurs during pregnancy. Maternal Pb can also be transferred to breastfeeding infants.

Malcoe et al. (2002) assessed Pb sources and their effect on blood Pb in rural Native American and white children living in a former mining region. Blood samples, residential environmental samples (soil, dust, paint, water), and caregiver interviews (hand-mouth behaviors, socioeconomic conditions) were obtained from a representative sample of 245 children ages 1–6 years. There were no ethnic differences in the results. However, poor children were especially vulnerable. Regression analysis showed that mean floor dust Pb loading >10.1 μg/ft^2 and yard soil Pb >165.3 mg/kg were independently associated with blood Pb levels ≥10 μg/dL.

The Pb content of dusts can be a significant source of exposure, especially for young children. Baseline estimates of potential human exposure to dusts, including intake due to normal hand-to-mouth activity, are 0.2 g/day for children 1–6 years old versus 0.1 g/day for adults when both indoor and outdoor ingestion of soil including dust is considered (EPA 1989c). For children who engage in pica behavior (the compulsive, habitual consumption of nonfood items), the ingestion rate of soil can be as high as 5 g/day. Although ingestion of Pb-containing paint may lead to elevated PbBs in young children, the major source of moderately elevated PbBs (30–80 μg/dL) in inner city children is most likely to be contaminated household dust and subsequent hand contamination and repetitive mouthing (Charney et al. 1980). Weathering of Pb-based paint can contribute to the Pb content of dust and soil. Pb levels of indoor dust and outdoor soil were found to be strongly predictive of PbBs in over 200 urban and suburban

infants followed from birth to 2 years of age; however, PbBs were not correlated with indoor air or tap water Pb levels, nor the size of nearby roadways. Indoor dust Pb levels and soil Pb levels in the homes of children with high PbBs (>8.8 µg/dL) were 72 µg/wipe (window sill dust) and 1,011 µg/g, respectively; children with low PbBs (<3.7 µg/dL) were exposed to 22 µg/wipe and 380 µg/g, respectively. In addition, 79% of the homes of children with high PbBs had been renovated, while only 56% of the homes of children with low PbBs had been renovated, suggesting that renovating the interior of homes previously painted with leaded paint may increase, at least temporarily, a child's exposure to Pb dust (Rabinowitz et al. 1985). Regular use of dust control methods (e.g., wet mopping of floors, damp-sponging of horizontal surfaces, high-efficiency vacuum cleaner) has been shown in some, although not all, cases to reduce indoor dust, Pb dust, and blood Pb levels in some, although not all, older homes containing leaded paints (Lanphear et al. 2000b; Rhoads et al. 1999). Decreases of between 17 and 43% in blood Pb concentrations were observed in children where regular dust control methods had been used to reduce indoor levels of Pb (Rhoads et al. 1999). EPA (2014c) summarized concentrations of Pb in house dust in the United Stated from 2006 to 2011; these data are presented in Table 5-24.

Table 5-24. Measurements of Lead in Indoor Dust in the United States from 2006–2011

Location	Sample site	Value reported
New York City, New York	Glass plate next to open window of academic building	Median weekly dust loading: 52 µg/m^2
Eureka, Utah near Eureka Mills Superfund Site	Indoor home site (not specified)	Dust concentrations, range: 160–2,000 mg/kg
Denver, Colorado, near Vasquez Blvd and I-70 Superfund Site	Indoor home site (not specified)	Dust concentrations, range: 11–660 mg/kg
East Helena, Montana, near East Helena Superfund Site	Indoor home site (not specified)	Dust concentrations, range: 68–1,000 mg/kg
Syracuse, New York	Floor	Dust concentrations, range: 209–1,770 mg/kg
United States (nationwide)	Smooth floor	Median dust loading: 1.7 µg/m^2 Average dust loading: 4.4 µg/m^2
	Rough floor	Median dust loading: 5.6 µg/m^2 Average dust loading: 16 µg/m^2
	Smooth windowsill	Median dust loading: 2.5 µg/m^2 Average dust loading: 190 µg/m^2
	Rough windowsill	Median dust loading: 55 µg/m^2 Average dust loading: 480 µg/m^2

Table 5-24. Measurements of Lead in Indoor Dust in the United States from 2006–2011

Location	Sample site	Value reported
Milwaukee, Wisconsin	Central perimeter	Average dust concentration: 107 µg/m^2
	Entry	Average dust concentration: 140 µg/m^2
	Window	Average dust concentration: 151 µg/m^2
Rural towns, Idaho	Vacuum	Dust concentration Median: 120 mg/kg Maximum: 830 mg/kg
	Floor	Median dust concentration: 95 mg/kg Maximum dust concentration: 1,300 mg/kg
Bunker Hill, Idaho Superfund Site	Vacuum	Median dust concentration: 470 mg/kg Maximum dust concentration: 2,000 mg/kg
	Floor	Median dust concentration: 290 mg/kg Maximum dust concentration: 4,600 mg/kg

Source: EPA 2014c

Lanphear and Roghmann (1997) and Lanphear et al. (1996a, 1996b, 1998b) studied factors affecting PbBs in urban children and found the following independent predictors of children's PbBs: dust Pb loading in homes (carpets, uncarpeted floors, window sills, and troughs), African-American race/ethnicity, foundation perimeter soil Pb levels, ingestion of soil or dirt, Pb content and condition of interior painted surfaces, and first-flush kitchen drinking water Pb levels (Lanphear et al. 1996a, 1996b). Differences in housing conditions and exposures to Pb-containing house dust appear to contribute to the racial differences in urban children's PbBs. In addition, white children were more likely to put soil in their mouths (outdoor exposure) and suck their fingers, and African-American children were more likely to put their mouths on window sills (indoor exposure) and to use a bottle. Exterior Pb exposures were more significant for white children and interior Pb exposures were more significant for African-American children (Lanphear et al. 1996a, 1996b). Mouthing behaviors are an important mechanism of Pb exposure among urban children (Lanphear and Roghmann 1997). Community characteristics such as residence within a city, proportion of African Americans, lower housing value, housing built before 1950, higher population density, higher rates of poverty, lower percent of high school graduates, and lower rates of owner-occupied housing have been used to identify children with elevated blood levels (Lanphear et al. 1998b). An analysis of children's PbBs and multiple measures of Pb concentrations in household dust, tap water, foundation perimeter soil, and interior house paint has been used to predict the effect of changing concentrations of Pb in environmental media on children's PbBs. An increase in dust Pb loading from background to 200 µg/ft^2 was estimated to produce an increase of 23.3% in the percentage of children estimated to have a PbB >10 µg/dL; an increase in tap water Pb concentration from

background to 15 µg/L was estimated to produce an increase of 13.7% in the percentage of children estimated to have a PbB level >10 µg/dL; and an increase in soil Pb concentration from background to 400 µg/g was estimated to produce an increase of 11.6% in the percentage of children estimated to have a PbB level >10 µg/dL (Lanphear et al. 1998a).

Outdoor Pb dust was found to be a more potent contaminant of children's hands than indoor dust at daycare centers in New Orleans; boys, in general, had higher hand Pb levels than girls. The conclusions were based on Pb analysis of hand wipe samples taken before and after children played outdoors at four different daycare centers (a private inner-city site, a private outer-city site, a public inner-city site, and a public outer-city site). The private inner-city site had a severely contaminated outdoor play area with measured soil Pb concentrations ranging from 287 to 1,878 mg/kg. The outdoor play area at the public inner-city site, where children exhibited the lowest hand Pb measurements of any site in the study, had been completely paved over with concrete or rubberized asphalt and had well-maintained equipment (Viverette et al. 1996).

EPA conducted the Urban Soil Lead Abatement Demonstration Project (USLADP), also known as the "Three City Lead Study," in Boston, Baltimore, and Cincinnati (EPA 1996c). The purpose was to determine whether abatement of Pb in soil could reduce PbBs of inner-city children. No significant evidence was found that soil abatement had any direct impact on children's PbBs in either the Baltimore or Cincinnati studies. In the Boston study, however, a mean soil Pb reduction of 1,856 ppm resulted in a mean decline of 1.28 µg/dL PbB at 11 months postabatement (Weitzman et al. 1993). Phase II extended the study to 2 years and included soil abatement of the two comparison areas from Phase I (Aschengrau et al. 1994). Combined results from Phase I and II suggested a higher impact of soil remediation on PbBs (2.2–2.7 µg/dL). EPA reanalyzed the data from the USLADP in an integrated report (EPA 1996c). They concluded that when soil is a significant source of Pb in the child's environment, under certain conditions, the abatement of that soil will result in a reduction in exposure and consequently, PbB level. The results of the USLADP suggest that a number of factors are important in determining the influence of soil remediation on PbBs in children. These include the site-specific exposure scenario, the magnitude of the remediation, and the magnitude of additional sources of Pb exposure.

Authors of a study of PbBs in children in Toronto, Canada, before and after abatement of Pb-contaminated soil and house dust found that they could neither strongly support nor refute beneficial effects of abatement. The failure to reach a definite conclusion from the results of the study, which included data from 12 cross-sectional blood-screening surveys that were conducted over an 8-year period,

was due, in part, to a low response rate (32–75%) to questionnaires used to determine behavioral, household, lifestyle, neighborhood, and environmental factors relating to study participants (Langlois et al. 1996).

Seasonal variations in PbBs in children have been observed in a number of studies (Gulson et al. 2008; Haley and Talbot 2004; Havlena et al. 2009; Kemp et al. 2007; Johnson and Bretsch 2002; Johnson et al. 1996; Laidlaw et al. 2005; Yiin et al. 2000). These studies suggest a general trend of increasing PbB during late summer and early fall. In addition to seasonal patterns in behavior (e.g., outdoor activities), seasonal patterns in weather (humidity and wind velocity) that promote re-entrainment and transport of dust Pb may contribute to the observed seasonal patterns in PbB (Laidlaw et al. 2005, 2012).

In addition to the ingestion of hand soil/dust through normal hand-to-mouth activity, some children engage in pica behavior (consumption of nonfood items), which can put them at increased risk through ingestion of large amounts of soil contaminated with Pb. It has been estimated that an average child may ingest between 20 and 50 mg of soil/day and that a pica child may ingest ≥5,000 mg of soil/day (LaGoy 1987; Mielke et al. 1989). If the soil contains 100 μg/g of Pb, an average child may be exposed to 5 μg Pb/day from this source alone (Mielke et al. 1989), and a pica child may be exposed to >100 times that amount.

Improper removal of Pb from housing known to contain Pb-based paint can significantly increase Pb levels in dust, thus causing Pb toxicity in children living in the home during the Pb-removal process. Four such cases have been documented (Amitai et al. 1987). In January 1995, the New York State Department of Health identified 320 children in 258 households in New York State (excluding New York City) with PbBs ≥20 μg/dL that were considered to be attributable to residential renovation and remodeling (CDC 1997a).

Workers occupationally exposed to Pb apparently carry Pb home on clothing, bodies, or tools. PbBs of children in households of occupationally exposed workers were almost twice those of children in neighboring homes whose parents were not occupationally exposed to Pb (median ranges were 10–14 and 5–8 μg/dL, respectively) (Grandjean and Bach 1986). Young children (<6 years old) of workers exposed to high levels of Pb in workplace air at an electronic components plant (61–1,700 μg Pb/m^3 ambient concentrations) had significantly elevated PbBs (13.4 μg/dL) compared with children from the same locale whose parents did not work in the electronics plant (7.1 μg/dL) (Kaye et al. 1987). Based upon data collected from 1987 to 1994, children aged 1–5 years (n=139) of workers whose occupation resulted

in Pb exposure had a geometric mean PbB of 9.3 μg/dL as compared to a U.S. population geometric mean of 3.6 μg/dL (Roscoe et al. 1999). Of this group, 52% of the children had PbBs ≥10 μg/dL compared to 8.9% of the U.S. population and 21% had PbBs ≥20 μg/dL compared to 1.1% of the U.S. population (Roscoe et al. 1999). Exposures of Pb workers' families have been identified in nearly 30 different industries and occupations. Industries in which exposure of family members has been reported most often include Pb smelting, battery manufacturing and recycling, radiator repair, electrical components manufacturing, pottery and ceramics, and stained glass making (NIOSH 1995). Children of Pb-exposed construction workers may also be at increased risk (Whelan et al. 1997).

Children may be exposed to Pb because of activities associated with certain hobbies and artistic activities practiced by adults in the home. Some of the more obvious hobbies and activities involving use of Pb-containing materials include casting, stained glass, pottery, painting, glassblowing, and screenprinting. Activities involving use of Pb-containing materials should always be done in an area well-ventilated with outdoor air and should never be done with children in the same room or in close proximity. Maas et al. (2005) indicated that high levels of Pb are prevalent in inexpensive cosmetic jewelry that is sold to the general public at retail stores.

Accidental or intentional ingestion of folk remedies (e.g., Chinese herbal medicines and Ayurvedic medicines containing Pb) or use of the Pb containing eye cosmetic tiro in children (discussed in Section 5.5.5) represents another source for potential Pb-poisoning in children. Hair dyes formulated with Pb acetate represent a potential source for Pb-poisoning both by accidental ingestion and by hand-to-mouth activity following contact with Pb-contaminated surfaces, including dyed hair of adults (Mielke et al. 1997).

Children may be exposed to Pb through the inhalation of second-hand smoke. Mannino et al. (2003) employed data from the NHANES III and analyzed PbBs of children aged 4–16 years who were exposed to high, low, and intermediate levels of second-hand smoke. Serum levels of the nicotine biomarker cotinine were used to classify the children into one of the three second-hand smoke exposure categories. The geometric mean PbBs were 1.5, 1.9, and 2.6 μg/dL for children with low (≤0.050–0.104 ng/mL), intermediate (0.105–0.562 ng/mL), and high (0.563–14.9 ng/mL) serum cotinine levels, respectively (Mannino et al. 2003).

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to workers exposed to Pb in the workplace, several other population groups at risk for potential exposure to high levels of Pb can be identified: preschool-age children and fetuses, individuals living near sites where Pb was produced or sites where Pb was disposed, and individuals living near one of the NPL hazardous waste sites where Pb has been detected in some environmental media (ATSDR 2015; EPA 1986b; Murgueytio et al. 1998) also may be at risk for exposure to high levels of Pb. Since Pb is often detected in tobacco and tobacco smoke, persons who use chewing tobacco or smoke or are exposed to second-hand smoke, may have higher PbB levels than persons that do not use these products (Apostolou et al. 2012; Bonanno et al. 2001; Richter et al. 2013). Recent studies have also found e-cigarettes to be a potential source of lead exposure (Olmedo et al. 2018).

General population exposure is most likely to occur through the ingestion of food and water contaminated with Pb; however, some individuals and families may be exposed to additional sources of Pb in their homes. This is particularly true of older homes that may contain Pb-based paint. In an attempt to reduce the amount of exposure due to deteriorating leaded paint, the paint is commonly removed from homes by burning (gas torch or hot air gun), scraping, or sanding. These activities have been found to result, at least temporarily, in higher levels of exposure for families residing in these homes. In addition, those individuals involved in the paint removal process (i.e., do-it-yourself renovators and professionals who remove Pb) can be exposed to such excessive levels that Pb poisoning may occur (Chisolm 1986; Fischbein et al. 1981; Rabinowitz et al. 1985). Special populations at risk of high exposure to tetraethyl Pb include workers at hazardous waste sites and those involved in the manufacture and dispensing of tetraethyl Pb (Bress and Bidanset 1991).

CHAPTER 6. ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of Pb is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the adverse health effects (and techniques for developing methods to determine such health effects) of Pb.

Data needs are defined as substance-specific informational needs that, if met, would reduce the uncertainties of human health risk assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.1 Information on Health Effects

Studies evaluating the health effects of exposure of humans Pb that are discussed in Chapter 2 are summarized in Figure 2-1. The purpose of this figure is to illustrate the information concerning the health effects of Pb. The number of human studies included in the profile for each endpoint is indicated regardless of whether an effect was found.

The health effects of Pb have been extensively studied in humans, including numerous studies in children. Due to the extent of the database in humans, a comprehensive review of the complete epidemiological database is not feasible. Epidemiological studies included in Chapter 2 were selected to identify the major lines of evidence regarding health effects in humans. Because the database of epidemiological studies is so large, animal studies were not included in the profile. Due to the increasing awareness that low-level environmental exposure resulting in blood Pb concentrations (PbB) <10 μg/dL is associated with adverse effects, particularly in children, the primary objective of current research is focused on health effects associated with PbB ≤10 μg/dL. Additional details on studies with PbB ≤10 μg/dL, including statistical analyses and assessment of confounding factors, are provided in the *Supporting Document for Epidemiological Studies for Lead.*

Health effects of Pb in humans are not defined in terms of route or duration of exposure. Epidemiological studies on Pb toxicity rely on internal exposure metrics (e.g., PbB), rather than measurements of external

exposures (e.g., concentration of Pb in water or air) or ingested dose. Furthermore, once absorbed into the body, the health effects of Pb are the same, regardless of the route of exposure. Environmental exposure to Pb occurs continuously over a lifetime and Pb can be retained in the body for decades; therefore, health effects of Pb in humans are considered to be associated with chronic exposure, rather than to shorter exposures.

6.2 Identification of Data Needs

A data need, as defined in ATSDR's *Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles* (ATSDR 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

Increased awareness of the potential adverse consequences of low environmental exposures to Pb has led to changes in U.S. public health policy, with a focus on lowering PbB levels to well below 10 μg/dL (CDC 2012d; EPA 2016b). In 2012, the CDC concluded that the 97.5th percentile of the U.S. PbB distribution (based on NHANES data) should be considered a reference value for identifying children who have "elevated" PbB (CDC 2012d). At that time, the reference was approximately 5 μg/dL, and the value continues to decline (NHANES 2011–2012; CDC 2018a). Therefore, additional epidemiological studies for all health outcomes are needed. The objective of these additional studies would be to define the low end of the dose-response curve (e.g., at PbB ≤5 μg/dL) and to identify threshold levels for health outcomes.

MRLs. Epidemiological studies have identified health effects of Pb in all organ systems. However, exposure thresholds for effects have not been identified, and it is not possible to determine from the epidemiological data which organ system are the most sensitive (i.e., primary) targets for Pb toxicity. Because clear thresholds for these effects have not been identified, MRLs for Pb have not been derived. Additional epidemiological studies would provide more data to further characterize effects; however, as PbBs continue to decline and effects are observed at the lowest PbB examined, identification of control groups has become increasingly difficult. Thus, it is not anticipated that additional epidemiological studies would identify threshold values for Pb-induced toxicity endpoints.

Health Effects. As noted above, epidemiological studies have identified health effects of Pb in every organ system at the lowest PbB evaluated. Additional prospective studies on all health outcomes would

provide important information to further characterize the effects of Pb and evaluate potential implications for long-term effects. However, as noted above, it is not anticipated that additional epidemiological studies would identify threshold values for health effects.

Epidemiology and Human Dosimetry Studies. Several models of the Pb exposure-biokinetics toxicokinetics in humans have been developed and used in dosimetry studies. Additional studies would be helpful for addressing major uncertainties in these models, including: (1) absence of calibration data for the kinetics of Pb in blood and bone in children in association with exposures that have been quantified with high certainty; (2) absence of calibration data on bone Pb concentrations in adolescents and adults in association with exposures that have been quantified with high certainty; (3) absence of data on the absolute bioavailability of ingested Pb in older children and adolescents; (4) incomplete understanding of Pb kinetics during periods of changing bone metabolism, including adolescence, pregnancy, and menopause; and (5) incomplete understanding of inter- and intra-individual variability in model parameters values in humans. In addition, there is a need for studies that can evaluate or validate model predictions of concentrations of Pb in blood and other tissues in populations in which PbBs are typical of the U.S. population ($\leq$5 μg/dL).

Biomarkers of Exposure and Effect. Measurement of blood Pb concentration is the most widely used biomarker of Pb exposure and is used to identity children who have elevated exposures. Measurement of bone Pb by XRF has been used to estimate Pb body burden in adults, which is a more accurate biomarker of long-term exposure than PbB. Additional studies that could improve and evaluate the validity of non-invasive biomarkers (e.g., hair, saliva, sweat, deciduous teeth, urine) for quantifying exposure would be helpful for population monitoring of Pb exposures and for epidemiology of Pb health effects.

Absorption, Distribution, Metabolism, and Excretion. Studies of Pb absorption are limited to studies in infants and adults. No data are available on the absorption of Pb in older children and adolescents. Additional studies of Pb absorption in this age category would be useful for improving exposure-biokinetic models.

A variety of factors are known to influence the absorption of ingested Pb, including the chemical form of the ingested Pb, the presence of food in the gastrointestinal tract, diet, and nutritional status with respect to calcium, vitamin D, and iron; however, for the most part, the mechanisms by which these interactions occur are not fully understood. This reflects, in part, a lack of understanding of the mechanisms by which

Pb is absorbed in the gastrointestinal tract, and studies aimed at elucidating such mechanisms would be helpful for developing PBPK models that accurately simulate relationships between Pb exposure and Pb in blood and other target and biomarker tissue.

The quantitative significance of the dermal absorption pathway as a contributor to Pb body burden remains an uncertainty. Few studies are available on Pb absorption after dermal exposure of inorganic Pb compounds in humans. Children may experience extensive dermal contact with Pb in soil, sand, or surface water and suspended sediment (e.g., beach or shoreline exposure scenario), even a low percent absorption across the skin may represent a significant internal dose. Therefore, additional studies designed to quantify dermal absorption of inorganic Pb compounds from both aqueous media and from soil would be helpful for improving PBPK models, in particular, studies that enable measurements to be extrapolated to children.

Comparative Toxicokinetics. Animal models (e.g., swine, mouse) have been used extensively as a model for assessing relative bioavailability of Pb in ingested soil in humans and for evaluating *in vitro* approaches to assessing bioaccessibility of Pb. However, no studies are available in which the absolute or relative bioavailability of ingested Pb has been quantitatively compared in animal models and humans. Such studies would be useful for validating both the *in vivo* swine model and the *in vitro* bioaccessibility model.

Children's Susceptibility. Children are likely to have increased susceptibility to Pb compared to adults for several reasons: increased susceptibility of developing physiological systems compared to mature systems; increased absorption of Pb in children compared to adults; and common childhood behaviors (e.g., hand-to-mouth activity, pica behavior [the compulsive, habitual consumption of nonfood items], proximity of breathing zone to entrained surface dust). In addition, several other factors may affect children's susceptibility to Pb, including (but not limited to) family socio-economic status, parent education, parent alcohol, tobacco, and drug use, allergen exposure, and family history of disease, although these factors may not be unique to children. Additional studies evaluating these factors would provide an increased understanding of relative contributions of these factors to child PbB and associated health effects.

Physical and Chemical Properties. No data needs were identified regarding physical and chemical properties of Pb.

Production, Import/Export, Use, Release, and Disposal. Continued monitoring of Pb production, import/export, use, release, and disposal would be helpful for identifying sources of potential human exposure. In particular, additional data on releases of Pb from leaded gasoline used in piston-driven engines would be helpful for determining potential contributions of this source to human exposure. Industrial wastes, as well as consumer products, containing Pb are disposed of in municipal and hazardous waste landfills. Current information on the amounts being disposed would be helpful for evaluating potential for exposures to Pb from these sources.

Environmental Fate. Additional information on the atmospheric transformations of organic and inorganic Pb compounds in the atmosphere would be helpful for identifying Pb compounds to which humans are most likely to be exposed by inhalation. Additional data regarding the chemical speciation and the transformation pathways of Pb in soils and water with varying properties such as pH, oxygen content, and salinity would be helpful for improving understanding of the environmental fate of Pb in soils and water.

Bioavailability from Environmental Media. Studies conducted in animal models show that oral RBA of soil Pb varies depending upon the Pb mineralogy and physical characteristics of the Pb in the soil. There is only one published study that assessed the bioavailability of Pb in humans (adults) who ingested hazardous waste site soil. Additional studies of this type would provide an improved basis for estimating Pb uptake in people who are exposed to Pb in soil. No studies have measured oral RBA of surface dusts. Since this is an important exposure pathway, especially in urban environments, studies of oral Pb RBA of surface dusts collected from various types of indoor and outdoor surfaces, including those impacted by paint Pb, would be helpful.

Recent interest in the use of soil-amending agents (e.g., phosphate) to reduce soil Pb bioavailability, would be served by additional studies directed at developing methods for monitoring the magnitude and persistence of the effect of amending agents on Pb bioavailability and for predicting the magnitude of the effect for improved design of amending projects.

Food Chain Bioaccumulation. No data needs were identified regarding food chain bioaccumulation.

Exposure Levels in Environmental Media. Reliable monitoring data for the levels of Pb in contaminated media at hazardous waste sites are needed so that the information obtained on levels of Pb in the environment can be used in combination with the known body burden of Pb to assess the potential

risk of adverse health effects in populations living in the vicinity of hazardous waste sites. Continued monitoring of Pb levels in air, drinking water, and diet (e.g., food and bottled water) would be helpful for evaluating potential for exposures to Pb from these sources. Continued testing of consumer products would be helpful for identifying potential localized sources of human exposure (e.g., ceramics, cosmetics, jewelry, toys).

Exposure Levels in Humans. Continued updating of national (e.g., NHANES) and regional surveys of Pb biomarkers (e.g., PbB) would be helpful for assessing temporal and demographic trends in Pb exposure in the U.S. population as well as for evaluating associations between Pb exposure and health metrics (e.g., those included in the NHANES), and for evaluating models that relate exposure to PbB.

Exposures of Children. Since an important variable in estimating Pb intakes from measurements of surface dust Pb levels is the rate of surface dust ingestion, improved estimates of soil ingestion would increase confidence in predictions of Pb intakes associated with exposures to Pb in surface dusts. In some contexts, exposure to surface dust Pb is measured in terms of Pb loading ($\mu g/Pb/cm^2$ of surface area available for contact); however, Pb loading measurements do not provide a direct way of estimating Pb ingestion without corresponding estimates of dust loading and surface dust ingestion rates. Improved methods for translating measurements of Pb loading into estimates of surface dust Pb concentration or surface dust Pb intake would be helpful for improving models for predicting exposure-Pb relationships in children.

6.3 Ongoing Studies

Ongoing studies on Pb are outlined in Table 6-1. Note that the studies listed below are funded by the National Institute of Health (NIH) and do not include ongoing studies that are funded by other sources.

Table 6-1. Ongoing Studies on Lead (Pb)

Investigator	Affiliation	Research description	Sponsor
D'Sa, VA	Memorial Hospital of Rhode Island	Longitudinal study on the relationship between PbB and myelination and neurite density in children ages 12–24 months	NICHD
Guilarte, TR	Florida International University	Mechanism of action study in rats to evaluate the role the NMDA receptor in Pb-induced neurotoxicity	NIEHS

Table 6-1. Ongoing Studies on Lead (Pb)

Investigator	Affiliation	Research description	Sponsor
Guilarte, TR	Florida International University	Study in rats to evaluate presynaptic mechanisms of Pb-induced neurotoxicity	NIEHS
Murphy, MP	University of Kentucky	Study in rats to evaluate mechanism of Pb-induced neurotoxicity	NIEHS
Sandler, DP	NIEHS	Epidemiological study examining Pb and other neurotoxins as risk factors for amyotrophic lateral sclerosis	NIEHS
Steenland, NK	Emory University	Epidemiological study examining mortality and renal disease in Pb-exposed workers	NIOSH
Bhattacharya, A	University of Cincinnati	Epidemiological study on the effects of early exposure to Pb as a risk for bone health later in life in African-American women	NIEHS
Wright, RO	Icahn School of Medicine Mount Sinai	Epidemiological study in children on the link between Pb exposure, stress, and neurological development	NIEHS

NICHD = National Institute of Child Health and Human Development; NIEHS = National Institute of Environmental Health Sciences; NIOSH = National Institute of Occupational Safety and Health; NMDA = N-methyl-D-aspartate; PbB = blood lead concentration

Source: NIH Reporter 2017

CHAPTER 7. REGULATIONS AND GUIDELINES

Pertinent international and national regulations, advisories, and guidelines regarding Pb in air, water, and other media are summarized in Table 7-1. This table is not an exhaustive list, and current regulations should be verified by the appropriate regulatory agency.

ATSDR develops MRLs, which are substance specific guidelines intended to serve as screening levels by ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. See 1.3 and APPENDIX A for information on procedures for deriving MRLs, as well as the deliberations and conclusions regarding derivation of MRLs for Pb.

Table 7-1. Regulations and Guidelines Applicable to Lead (Pb)

Agency	Description	Information	Reference
	Air		
EPA	RfC	No data	IRIS 2002, 2004
EPA	NAAQS	0.15 µg/m^3 [a]	EPA 2016e
WHO	Air quality guidelines	No data	WHO 2010
	Water & Food		
EPA	Drinking water standards and health advisories	No data	EPA 2012
	National primary drinking water regulations for inorganic lead		EPA 2009
	TT	TT5	
	Action level	0.015 mg/L [b]	
	Public health goal	zero	
	RfD		
	Tetraethyl lead	1 x10^{-7} mg/kg/day [c]	IRIS 2002
WHO	Drinking water quality guidelines		WHO 2017
	Provisional guideline value, lead	0.01 mg/L (10 µg/L) [d]	
FDA	EAFUS [e]	No data	FDA 2013
	Cancer		
ACGIH	Carcinogenicity classification		
	Lead (inorganic compounds as Pb)	A3 [f,g]	ACGIH 2001a, 2016
	Lead chromate (as Pb or chromium)	A2 [h,i]	ACGIH 2001b, 2016
	Tetraethyl lead	A4 [j,k]	ACGIH 2001c, 2016
HHS	Carcinogenicity classification		NTP 2016
	Lead and lead compounds	Reasonably anticipated to be human carcinogens [l]	

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Table 7-1. Regulations and Guidelines Applicable to Lead (Pb)

Agency	Description	Information	Reference
EPA	Carcinogenicity classification		IRIS 2004
	Lead and compounds (inorganic)	B2[m,n]	
IARC	Carcinogenicity classification		
	Lead	Group 2B[o,p]	IARC 1987, 2017
	Lead compounds, inorganic	Group 2A[q,r]	IARC 2006, 2017
	Lead compounds, organic	Group 3[s,t]	IARC 2006, 2017
	Occupational		
ACGIH	TLV		ACGIH 2016
	Lead	0.05 mg/m^3	
	Lead chromate (as Pb)	0.05 mg/m^3	
	Lead chromate (as chromium)	0.012 mg/m^3	
	Tetraethyl lead	0.1 mg/m^3	
	BEI		
	Lead in blood	30 µg/100 mL[u]	
OSHA	PEL (8-hour TWA) for general industry		
	Lead (elemental, inorganic and organic soaps)	50 µg/m^3	OSHA 2016a
	Tetraethyl lead and tetramethyl lead	0.075 mg/m^3 [v]	OSHA 2016b
	PEL (8-hour TWA) for construction and shipyards		
	Lead (elemental, inorganic and organic soaps)	50 µg/m^3	OSHA 2016c, 2016d
	Tetraethyl lead	0.1 mg/m^3 [v]	OSHA 2016e, 2016f
	Tetramethyl lead	0.15 mg/m^3 [v]	OSHA 2016e, 2016f
	Action level (8-hour TWA) for general industry, construction		
	Lead (elemental, inorganic and organic soaps)	30 µg/m^3	OSHA 2016a, 2016c
NIOSH	REL (8-hour TWA)		
	Lead and compounds (as Pb)	0.05 mg/m^3	NIOSH 2016b
	Tetraethyl lead (as Pb)	0.075 mg/m^3 [v]	NIOSH 2016c
	IDLH		
	Lead and compounds (as Pb)	100 mg/m^3	NIOSH 2016b
	Tetraethyl lead (as Pb)	40 mg/m^3	NIOSH 2016c
	Emergency Criteria		
EPA	AEGLs-air	No data	EPA 2016d
DOE	PACs-air		DOE 2016
	Lead		
	PAC-1	0.15 mg/m^3	
	PAC-2	120 mg/m^3	
	PAC-3	700 mg/m^3	

Table 7-1. Regulations and Guidelines Applicable to Lead (Pb)

Agency	Description	Information	Reference
	Tetraethyl lead		
	PAC-1	0.5 mg/m^3	
	PAC-2	5.5 mg/m^3	
	PAC-3	33 mg/m^3	
	Lead acetate		
	PAC-1	5 mg/m^3	
	PAC-2	55 mg/m^3	
	PAC-3	330 mg/m^3	
	Lead carbonate		
	PAC-1	0.19 mg/m^3	
	PAC-2	24 mg/m^3	
	PAC-3	900 mg/m^3	
	Lead dioxide and lead sulfide		
	PAC-1	0.17 mg/m^3	
	PAC-2	140 mg/m^3	
	PAC-3	810 mg/m^3	
	Lead tetroxide		
	PAC-1	0.17 mg/m^3	
	PAC-2	130 mg/m^3	
	PAC-3	770 mg/m^3	
	Lead oxide		
	PAC-1	0.16 mg/m^3	
	PAC-2	130 mg/m^3	
	PAC-3	750 mg/m^3	
	Lead sulfate		
	PAC-1	0.22 mg/m^3	
	PAC-2	170 mg/m^3	
	PAC-3	1,000 mg/m^3	
	Lead phosphate		
	PAC-1	0.2 mg/m^3	
	PAC-2	150 mg/m^3	
	PAC-3	910 mg/m^3	
	Lead chloride		
	PAC-1	0.2 mg/m^3	
	PAC-2	160 mg/m^3	
	PAC-3	940 mg/m^3	
	Lead chromate		
	PAC-1	0.036 mg/m^3	
	PAC-2	16 mg/m^3	
	PAC-3	97 mg/m^3	

Table 7-1. Regulations and Guidelines Applicable to Lead (Pb)

Agency	Description	Information	Reference
	Lead bromide		
	PAC-1	0.27 mg/m^3	
	PAC-2	200 mg/m^3	
	PAC-3	1,200 mg/m^3	
	Lead nitrate		
	PAC-1	0.24 mg/m^3	
	PAC-2	180 mg/m^3	
	PAC-3	1,100 mg/m^3	
	Lead iodide		
	PAC-1	0.33 mg/m^3	
	PAC-2	270 mg/m^3	
	PAC-3	1,600 mg/m^3	
	Lead fluoroborate		
	PAC-1	0.28 mg/m^3	
	PAC-2	220 mg/m^3	
	PAC-3	1,300 mg/m^3	
	Miscellaneous Federal Guidance		
CDC	PbB reference value	5 µg/dL[w]	CDC 2012d, 2012f
EPA	Soil screening level	400 ppm[x]	EPA 1994e, 1998; 2016f
HUD	Dust lead hazard action levels[y]		HUD 2017
	Floors	≥10 µg/ft^2	
	Window sills	≥100 µg/ft^2	
	Dust lead clearance action levels[y]		
	Interior floors	<10 µg/ft^2	
	Porch floors	<40 µg/ft^2	
	Window sills	<100 µg/ft^2	
	Window troughs	<100 µg/ft^2	

[a]Not-to-exceed air Pb concentration of 0.15 µg/m^3 in total suspended solids for a 3-month rolling average, evaluated over a 3-year period (i.e., the 3-month rolling average cannot exceed 0.15 µg/m^3 over a 3-year period). Based on a variety of lines of evidence including human epidemiological evidence for a variety of adverse health effects in children and other at-risk populations, most notably effects on the developing nervous system.
[b]Potential health effects from long-term exposure above the MCL include delays in physical or mental development in infants and children (children could show slight deficits in attention span and learning abilities) and kidney problems and high blood pressure in adults.
[c]Based on histopathology of liver and thymus; POD = LOAEL: 1.2x10^{-3} mg/kg/day; composite uncertainty factor=10,000; confidence=medium.
[d]The guideline value is designated as provisional on the basis of treatment performance and analytical achievability because it is extremely difficult to achieve a lower concentration by central conditioning, such as phosphate dosing.
[e]The EAFUS list of substances contains ingredients added directly to food that FDA has either approved as food additives or listed or affirmed as GRAS.
[f]A3: confirmed animal carcinogen with unknown relevance to humans.
[g]Based on demonstrated carcinogenicity of soluble lead compounds in animals.
[h]A2: Suspected human carcinogen.
[i]Based on experimental animal and epidemiological studies that demonstrated a low degree of carcinogenicity.
[j]A4: not classifiable as a human carcinogen.
[k]Based on inconclusive evidence of carcinogenicity in rodent bioassays.
[l]Based on sufficient evidence of carcinogenicity in experimental animals.

Table 7-1. Regulations and Guidelines Applicable to Lead (Pb)

Agency	Description	Information	Reference

[m]Group B2: probable human carcinogen.
[n]Based on sufficient evidence of carcinogenicity in animals.
[o]Group 2B: possibly carcinogenic to humans.
[p]Based on inadequate evidence for carcinogenicity to humans and sufficient evidence for carcinogenicity of inorganic lead compounds in animals.
[q]Group 2A: probably carcinogenic to humans.
[r]Based on limited evidence of carcinogenicity of inorganic lead in humans and sufficient evidence of carcinogenicity in experimental animals. Also based on sufficient evidence of carcinogenicity of inorganic compounds in experimental animals, sufficient evidence of carcinogenicity of Pb acetate, Pb subacetate, Pb chromate, and Pb phosphate in experimental animals, and based on inadequate evidence of carcinogenicity of Pb oxide, Pb arsenate, tetraethyl Pb, and Pb powder in experimental animals.
[s]Group 3: not classifiable as to carcinogenicity to humans.
[t]Based on inadequate evidence of carcinogenicity in humans and inadequate evidence of carcinogenicity in experimental animals.
[u]2016 Notice of Intended Change for BEI: 200 µg/L.
[v]Skin designation.
[w]Based on 97.5th percentile of NHANES PbB distribution in children 1-5 years of age.
[x]Based on evidence for adverse effects on the developing nervous system in association with PbB in the 2–8 µg/dL range.
[y]Based on evidence for adverse effects on the developing nervous system in association with PbB <5 µg/dL.

ACGIH = American Conference of Governmental Industrial Hygienists; AEGL = acute exposure guideline levels; AIHA = American Industrial Hygiene Association; BEI = biological exposure index; CDC = Centers for Disease Control and Prevention; DOE = Department of Energy; EAFUS = Everything Added to Food in the United States; EPA = Environmental Protection Agency; ERPG = emergency response planning guidelines; FDA = Food and Drug Administration; GRAS = generally recognized as safe; HHS = Department of Health and Human Services; HUD = Housing and Urban Development; IARC = International Agency for Research on Cancer; IDLH = immediately dangerous to life or health concentration; IRIS = Integrated Risk Information System; LOAEL = lowest-observed-adverse-effect level; MCL = maximum contaminant level; NAAQS = National Ambient Air Quality Standard; NIOSH = National Institute for Occupational Safety and Health; NTP = National Toxicology Program; OSHA = Occupational Safety and Health Administration; PAC = Protective Action Criteria; PEL = permissible exposure limit; POD = point of departure; REL = recommended exposure limit; RfC = inhalation reference concentration; RfD = oral reference dose; TLV = threshold limit values; TT = treatment technique; TWA = time-weighted average; WHO = World Health Organization